



King's Research Portal

DOI:

[10.1097/PSY.0000000000000388](https://doi.org/10.1097/PSY.0000000000000388)

Document Version

Peer reviewed version

[Link to publication record in King's Research Portal](#)

Citation for published version (APA):

Baldwin, J. R., Arseneault, L., Odgers, C., Belsky, D. W., Matthews, T., Ambler, A. P., Caspi, A., Moffitt, T. E., & Danese, A. (2016). Childhood Bullying Victimization and Overweight in Young Adulthood: A Cohort Study. *Psychosomatic Medicine*, 78(9), 1094-1103. <https://doi.org/10.1097/PSY.0000000000000388>

Citing this paper

Please note that where the full-text provided on King's Research Portal is the Author Accepted Manuscript or Post-Print version this may differ from the final Published version. If citing, it is advised that you check and use the publisher's definitive version for pagination, volume/issue, and date of publication details. And where the final published version is provided on the Research Portal, if citing you are again advised to check the publisher's website for any subsequent corrections.

General rights

Copyright and moral rights for the publications made accessible in the Research Portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognize and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the Research Portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the Research Portal

Take down policy

If you believe that this document breaches copyright please contact librarypure@kcl.ac.uk providing details, and we will remove access to the work immediately and investigate your claim.

**CHILDHOOD BULLYING VICTIMIZATION AND SUBSEQUENT OVERWEIGHT IN
YOUNG ADULTHOOD: A COHORT STUDY**

Jessie R. Baldwin MSc¹, Louise Arseneault PhD¹, Candice Odgers PhD^{2,3,4}, Daniel W. Belsky PhD^{5,6}, Timothy Matthews BSc¹, Antony Ambler MSc¹, Avshalom Caspi PhD^{1,4,7}, Terrie E. Moffitt PhD^{1,4,7}, and Andrea Danese MD PhD^{1,8,9}

Author Affiliations:

¹MRC Social, Genetic and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK

²Center for Child and Family Policy, Duke University, Durham, NC, USA

³Sanford School of Public Policy, Duke University, Durham, NC, USA

⁴Department of Psychology and Neuroscience, Duke University, Durham, NC, USA

⁵Social Science Research Institute, Duke University, Durham, North Carolina.

⁶Department of Medicine, Duke University School of Medicine, Durham, NC, USA

⁷Department of Psychiatry and Behavioral Sciences, Duke University, Durham, NC, USA

⁸Department of Child and Adolescent Psychiatry, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK

⁹National and Specialist Clinic for Child Traumatic Stress and Anxiety Disorders, South London and Maudsley NHS Foundation Trust, London, UK

Correspondence: Dr Andrea Danese, Social, Genetic and Developmental Psychiatry Centre (MRC), Institute of Psychiatry, Psychology and Neuroscience, De Crespigny Park, Denmark Hill, London, United Kingdom, SE5 8AF. Tel: +442078480601. Fax: +442078480866, Email: andrea.danese@kcl.ac.uk

Conflicts of Interest and Source of Funding: No conflicts of interest declared. The E-Risk Study is funded by the Medical Research Council (UKMRC) Grant G1002190. Additional support was provided by National Institute of Child Health and Development Grants (HD077482 and HD061298), Economic and Social Research Council Grant RES-177-25-0013, the Jacobs Foundation, the Avielle Foundation, and by a NARSAD Young Investigator Award to Andrea Danese. Jessie Baldwin is funded by the Economic and Social Research Council.

6,500 WORDS, 3 TABLES, 1 FIGURE

ABSTRACT

Objective

To test whether bullied children have an elevated risk of being overweight in young adulthood, and whether this association is: (1) consistent with a dose-response relationship - namely its strength increases with the chronicity of victimization; (2) consistent across different measures of overweight; (3) specific to bullying and not explained by co-occurring maltreatment; (4) independent of key potential confounders; and (5) consistent with the temporal sequence of bullying preceding overweight.

Method

A representative birth cohort of 2,232 children was followed to age 18 years as part of the Environmental Risk (E-Risk) Longitudinal Twin Study. Childhood bullying victimization was reported by mothers and children during primary school and early secondary school. At age 18, we assessed a categorical measure of overweight, body mass index (BMI), and waist-hip ratio. Indicators of overweight were also collected at ages 10 and 12. Co-twin body-mass and birth weight were used to index genetic and fetal liability to overweight, respectively.

Results

Bullied children were more likely to be overweight than non-bullied children at age 18, and this association was: (1) strongest in chronically bullied children (OR=1.69, 95% CI=1.21-2.35); (2) consistent across measures of overweight (BMI: $b=1.12$, 95% CI=0.37-1.87; waist-hip ratio: $b=1.76$, 95% CI=0.84-2.69); (3) specific to bullying and not explained by co-occurring maltreatment; (4) independent of socio-economic status, food insecurity, child mental health/cognition, and pubertal development; and (5) not present at the time of bullying victimization, and independent of childhood weight and genetic and fetal liability.

Conclusion

Childhood bullying victimization predicts overweight in young adulthood.

KEYWORDS: Bullying, victimization, early life stress, overweight, longitudinal study

CHILDHOOD BULLYING VICTIMIZATION AND SUBSEQUENT OVERWEIGHT IN YOUNG ADULTHOOD: A COHORT STUDY

Overweight affects 69% of adults in the United States (1), increases risk of cardiovascular disease, Type-2 diabetes, and cancer (2), and is associated with social discrimination (3). There is little evidence that readily available interventions targeting overweight, such as diet and behavioral changes, have long-term effectiveness (4). Therefore, it is important to identify potentially modifiable risk factors as targets for prevention.

Experiences during sensitive childhood periods may have long-lasting effects on body mass (5). Experimental evidence from non-human primates shows that chronic psychosocial stress in early life can lead to greater body mass in later life (6, 7). Similarly, observational studies of humans suggest that early life stress can predispose to excess body mass. For example, individuals with a history of childhood maltreatment have an elevated risk of obesity in adulthood (8) and show faster gains in body mass index (BMI) over their lifetime (9) compared to non-maltreated individuals. However, maltreatment by adults is only one of several prevalent, chronic, and severe childhood stressors. To test the broader hypothesis that early life stress predisposes to excess body mass in humans, it is important to test whether findings related to maltreatment generalize to other such stressors.

Childhood bullying victimization is another severe stressor increasingly targeted by public health campaigns (10, 11). Emerging evidence suggests that bullying victimization is associated with overweight in later life (12-14). Despite these initial findings, several outstanding questions remain. First, it is important to establish whether the association between bullying victimization and overweight is consistent with a *dose-response relationship*, with greater chronicity of exposure predicting greater risk of becoming overweight. Yet, it is unclear if overweight risk is a function of the chronicity of bullying

victimization. Second, it is important to test whether the association between bullying victimization and overweight is *consistent* across different measures of overweight. However, it is unclear if the association generalizes from global measures like BMI to measures of central adiposity, such as waist-hip ratio, which predict disease risks over and above BMI (15). Third, it is important to establish whether the association with overweight risk is *specific* to bullying victimization. Bullying victimization often co-occurs with childhood maltreatment (16), and it is unclear whether maltreatment accounts for the association. Fourth, it is important to test whether the association between bullying victimization and overweight is *independent* of confounding linked to psychosocial risk or child characteristics. For example, bullying victimization occurs more frequently in the context of socioeconomic disadvantage and food insecurity (16, 17), which are risk factors for overweight (18). Additionally, children with externalizing problems, internalizing problems, and low IQ are more liable to bullying victimization (16), as well as later overweight (14). Furthermore, early pubertal development is associated with bullying victimization (19) and predicts overweight (20). However, it is unclear if co-occurring psychosocial risks or child characteristics confound the association. Finally, because overweight children may be more likely to be bullied (13, 21), it is important to test whether bullying victimization *precedes* the development of overweight. However, it is unclear if the association between childhood bullying victimization and later overweight is independent of continuity in body mass or of genetic and fetal liability (22, 23). We sought to test these key questions in a birth cohort of 2,232 twins followed to age 18.

Method

Study sample

Participants were members of the Environmental Risk (E-Risk) Longitudinal Twin Study, which tracks the development of a birth cohort of 2,232 British children. The sample was drawn from a larger birth register of twins born in England and Wales in 1994-95 (24). Full details about the sample are reported elsewhere (25). Briefly, the E-Risk sample was

constructed in 1999-2000, when 1,116 families (93% of those eligible) with same-sex 5-year-old twins participated in home-visit assessments. This sample comprised 55% monozygotic (MZ) and 45% dizygotic (DZ) twin pairs; sex was evenly distributed within zygosity (49% male). Seven percent of the study members self-identified as Black, Asian, or mixed-race. Families were recruited to represent the U.K. population of families with newborns in the 1990s, on the basis of residential location throughout England and Wales and mother's age. Teenaged mothers with twins were over-selected to replace high-risk families who were selectively lost to the register through non-response. Older mothers having twins via assisted reproduction were under-selected to avoid an excess of well-educated older mothers.

At follow up, the study sample represents the full range of socioeconomic conditions in the U.K., as reflected in the families' distribution on a neighborhood-level socioeconomic index (ACORN [A Classification of Residential Neighbourhoods], developed by CACI Inc. for commercial use in Great Britain)(26). ACORN uses census and other survey-based geodemographic discriminators to classify enumeration districts (~150 households) into socioeconomic groups ranging from "wealthy achievers" (Category 1) with high incomes, large single-family houses, and access to many amenities, to "hard pressed" neighborhoods (Category 5) dominated by government-subsidized housing estates, low incomes, high unemployment, and single parents. ACORN classifications were geocoded to match the location of each E-Risk study family's home (27). E-Risk families' ACORN distribution closely matches that of households nation-wide: 25.6% of E-Risk families live in "wealthy achiever" neighborhoods compared to 25.3% nationwide; 5.3% vs. 11.6% live in "urban prosperity" neighborhoods; 29.6% vs. 26.9% live in "comfortably off" neighborhoods; 13.4% vs. 13.9% live in "moderate means" neighborhoods; and 26.1% vs. 20.7% live in "hard-pressed" neighborhoods. E-Risk underrepresents "urban prosperity" neighborhoods because such households are likely to be childless.

Follow-up home visits were conducted when children were aged 7 (98% participation), 10 (96% participation), 12 (96% participation), and, in 2012-2014, at 18 years (93% participation). There were 2,066 children who participated in the E-Risk assessments at age 18, comprising 55% MZ and 45% DZ twin pairs, with a reasonably even split between genders (47% male). The average age of the twins at the time of the assessment was 18.4 years (SD = 0.36); all interviews were conducted after the 18th birthday. There were no differences between those who did and did not take part at age 18 in terms of socioeconomic status (SES) assessed when the cohort was initially defined ($X^2 = 0.86$, $p = 0.65$), age 5 IQ scores ($t = 0.98$, $p = 0.33$), age 5 internalizing or externalizing behaviour problems ($t = 0.40$, $p = 0.69$ and $t = 0.41$, $p = 0.68$, respectively), childhood bullying victimization ($X^2 = 0.57$, $p = 0.75$), and age-10 or age-12 weight ratings ($t = -1.40$, $p = 0.16$ and $t = -.98$, $p = 0.33$, respectively). Home visits at ages 5, 7, 10, and 12 years included assessments with participants as well as their mother (or primary caretaker); the home visit at age 18 included interviews only with the participants. Each twin participant was assessed by a different interviewer.

The Joint South London and Maudsley and the Institute of Psychiatry Research Ethics Committee approved each phase of the study. Parents gave informed consent and twins gave assent between 5-12 years and then informed consent at age 18.

Bullying victimization

We assessed experiences of victimization by bullies using both mothers' and children's reports of victimization at primary and secondary school (28). We explained, "Someone is being bullied when another child (a) says mean and hurtful things, makes fun, or calls a person mean and hurtful names; (b) completely ignores or excludes someone from their group of friends or leaves them out on purpose; (c) hits, kicks, or shoves a person, or locks

them in a room; (d) tells lies or spreads rumours about them; and (e) other hurtful things like these. We call it bullying when these things happen often, and when it is difficult to make it stop. We do not call it bullying when it is done in a friendly or playful way.” Mothers were interviewed when children were 7, 10, and 12 years old and asked whether either twin had been bullied by another child, responding *never, yes, or frequently*. We combined mothers’ reports from the age 7 and 10 assessments to derive a measure of victimization during primary school. Mothers’ reports at the age 12 assessment indexed victimization during secondary school. During private interviews with children when they were 12 years old, they indicated whether they had been bullied by another child during primary or secondary school. Typically, relatively low levels of cross-informant agreement for bullying involvement are observed (29, 30). In keeping with other studies, the cross-informant agreement between mother and child reports of victimization during primary school and secondary school were modest: $k = 0.20$ during primary school and $k = 0.29$ during secondary school. Although agreement between mothers and children was only modest, reports of victimization from both informants were similarly associated with children’s internalizing and externalizing problems, suggesting that each informant provides a unique but meaningful perspective on bullying victimization (31). The test–retest reliability of victimization was 0.87 using a sample of 30 parents who were interviewed twice, 3–6 weeks apart. When a mother or a child reported victimization, the interviewer asked them to describe what happened. Notes taken by the interviewers were later checked by an independent rater to verify that the events reported could be classified as instances of bullying operationally defined as evidence of (a) repeated harmful actions (b) between children (c) where there is a power differential between the bully and the victim (31). We summed mother and child reports of victimization across primary school and separately across secondary school to capture all instances of victimization during these two periods. As data were positively skewed for both the primary and secondary school measures, we divided each index of victimization into three category variables: (0) never victimized (primary school: $N=872$, 39.4%; secondary school: $N=1,138$, 53.0%), (1) reported by either mother or child as being occasionally victimized (primary

school: N=646, 29.2%; secondary school: N=517, 24.1%), and (2) reported as being victimized by both informants, or as frequently victimized by mother or child (primary school: N=696, 31.4%; secondary school: N=491, 22.9%). From this information, we derived a measure of chronic bullying victimization across primary and early secondary school encompassing exposure over the childhood years. The sample was divided into three groups: (0) non-victims (children who experienced either occasional or no victimization at primary and secondary school; N=1,255, 58.5%), (1) transitory victims (frequently victimized at primary school only or secondary school only; N=605, 28.2%), and (2) chronic victims (frequently victimized at both primary and secondary school; N=286, 13.3%).

Overweight

Measures of overweight in young adulthood. Trained research workers took anthropometric measurements of study members when they were aged 18 years. BMI was computed as weight in kilograms over squared height in meters. Waist-hip ratio was calculated by dividing waist circumference by hip circumference. Overweight was defined according to US Centers for Disease Control and Prevention Criteria according to age- and sex-specific growth charts (32). Study members with a BMI equal to or above the 85th percentile were classified as overweight.

Measures of overweight in childhood. At the age 10 and 12 assessments, research workers rated children's weights on a 7-point scale (with 1 being underweight and 7 being overweight). These ratings were based on visual assessment, with the rationale that victims of violence might be targeted because of the perpetrator's own visual assessment. Research worker ratings of weight at age 10 were correlated with their ratings at age 12 ($r = 0.58$). At age 12, research workers also took anthropometric measurements in a subsample of study members (N=173). Measured BMI in this subsample at age 12 was

correlated with research worker ratings of weight at age 12 ($r = 0.59$) and ratings of weight at age 10 ($r = 0.45$).

Covariates

Childhood maltreatment. Methods used to assess childhood maltreatment in our sample have been described in detail elsewhere (33, 34). We assessed physical maltreatment by an adult using a standardized clinical interview protocol (35) designed to enhance mothers' comfort with reporting valid child maltreatment information, while also meeting researchers' responsibilities for referral under the UK Children Act. No family has left the study after intervention. When mothers reported any maltreatment, interviewers followed with standardized probes (for example, accidental harm was ruled out; harm by peers was coded as bullying, not maltreatment). Sexual abuse was queried directly. Over the years of data collection, the study maintained a cumulative dossier for each child, composed of recorded debriefings with interviewers who had coded any indication of maltreatment at any of the four successive home visits, recorded narratives of the four successive caregiver interviews at child ages 5, 7, 10 and 12 years (covering the period from birth to 12 years), and information from clinicians whenever the study made a referral. On the basis of the review of each child's cumulative dossier, two clinical psychologists (Professor Terrie E Moffitt and the project coordinator) reached consensus for whether physical maltreatment had occurred. Examples of maltreatment included the following: the mother smacked the child weekly, leaving marks or bruises; child was repeatedly beaten by a young adult stepsibling; child was routinely smacked by father when drunk, 'just to humiliate him'; child was fondled sexually and often slapped by the mother's boyfriend. Many, but not all, cases identified in the course of our research were under investigation by the police or social services, already on the child protection register, or in foster care at follow-up, having been taken away from their parents because of abuse. On the basis of the mother's report of the severity of maltreatment and the interviewer's rating of the likelihood that the child had been physically

maltreated, children were coded as having experienced no maltreatment (N=1,760, 78.9%), probable maltreatment (N=344, 15.4%) or definite maltreatment (N=128, 5.7%).

Socioeconomic status. The family socioeconomic status at the age of 5 years was defined through a standardized composite of parental income, education and occupation. The three socioeconomic status indicators were highly correlated ($r = 0.57-0.67$) and loaded significantly onto one latent factor (36). The population-wide distribution of the resulting factor was divided in tertiles for analyses.

Food insecurity. History of food insecurity was reported by the mother to a clinical interviewer when children were aged 7 and 10 years using a seven-item scale developed by the US Department of Agriculture (37). Using data from both assessments, we classified families as having experienced no food insecurity (N=1914, 87.1%), episodic food insecurity (if food insecurity was reported at age 7 or age 10 assessments; N=210, 9.6%) or sustained food insecurity (food insecurity at both age 7 and age 10 assessments; N=74, 3.4%).

Child mental health/cognition. We assessed internalizing and externalizing problems at age 5 by using the Child Behavior Checklist in face-to-face interviews with mothers and requesting the teacher's report for each child (38, 39). The internalizing problems scale is the sum of items in the withdrawn and anxious/depressed subscales, and the externalizing problems scale is the sum of items from the aggressive and delinquent subscales. We summed and standardised mothers' and teachers' reports to create cross-informant scales. We tested children's IQ at age 5 individually by using a short form of the Wechsler Preschool and Primary Scale of Intelligence—Revised (40, 41).

Pubertal development. Pubertal maturation at age 12 was evaluated through maternal

ratings of Tanner's stages (42) during home visits. Sex-specific variables were combined to obtain an overall index of pubertal maturation for each study member.

Genetic risk of overweight. Genetic risk was calculated according to co-twin zygosity and overweight status according to a method used previously (43, 44), with coding of 0 for the monozygotic co-twin of a non-overweight twin (lowest risk), 1 for the dizygotic co-twin of a non-overweight twin, 2 for the dizygotic co-twin of an overweight twin, and 3 for the monozygotic co-twin of an overweight twin (highest risk).

Birth weight. Each twin's birth weight was obtained by means of parental recall when the twins were 1 year old (45).

Statistical analyses

First, we tested whether the chronicity of childhood bullying victimization predicted being overweight at age 18 in a logistic regression model. Second, we tested whether the association between childhood bullying victimization and overweight at age 18 generalized to continuous measures of BMI and waist-hip ratio at age 18, in linear regression models. Third, we tested whether the association between childhood bullying victimization and overweight at age 18 in the above models was accounted for by child maltreatment. Fourth, we tested whether the association between childhood bullying victimization and overweight at age 18 was accounted for by socio-economic status and food insecurity, child mental health/cognition, and pubertal development. Finally, we tested whether childhood bullying victimization preceded overweight, by (1) testing whether bullied children were overweight at ages 10 and 12, and (2) testing whether childhood bullying victimization predicted overweight at age 18 after accounting for (i) weight at ages 10 and 12, and (ii) genetic risk of overweight and birth weight. We controlled for the effects of gender and ethnicity in all multivariate analyses. To correct for the inclusion of two study children in each family, we

adjusted all analyses for the effect of familial clustering (using the option *cluster* in STATA SE, 13th edition). Pregnant women were removed from all analyses.

Results

Is the association between childhood bullying victimization and overweight at age 18 influenced by the chronicity of exposure?

Bullied children were more likely to be overweight at age 18 than non-bullied children (Table 1; Figure 1). The risk of being overweight increased as a function of the chronicity of bullying victimization, with children bullied in both primary school and secondary school showing the highest risk of being overweight (Table 2- baseline model). Effects were similar in boys and girls (sex-interaction term p -value=0.41).

Is the association between bullying victimization and overweight at age 18 consistent across different measures?

Bullied children also showed higher BMI and waist-hip ratio at age 18 than non-bullied children (Table 1; Figure 1). The association between bullying victimization and BMI was only seen in chronically victimized children, whereas children who experienced either transitory or chronic victimization had a greater waist-hip ratio than controls (Table 2- baseline model). Again, these effects were similar in boys and girls (sex-interaction terms p -value=0.23 for BMI and p =0.53 for waist-hip ratio).

Is the association between bullying victimization and overweight at age 18 explained by co-occurring maltreatment?

Bullied children were more likely to have experienced maltreatment than non-bullied children

(Table 1). In turn, child maltreatment predicted higher waist-hip ratio at age 18 (Table 3) and an elevated risk of being overweight in females (definite maltreatment: OR=2.18, 95% CI=1.07-4.45), but not in males (definite maltreatment: OR=1.04, 95% CI=-0.49-2.20). However, even after accounting for maltreatment by an adult, bullied children were more likely to be overweight at age 18 than non-bullied children (Table 2- model 1).

Is the association between bullying victimization and overweight at age 18 independent of confounding by psychosocial risks and child characteristics?

Bullying victimization was associated with psychosocial risk factors (socioeconomic disadvantage and food insecurity), poor childhood mental health/cognition (externalizing problems, internalizing problems, and low IQ), and early pubertal development (Table 1). With the exception of child internalizing problems, these variables all predicted overweight at age 18 (Table 3). However, bullied children showed an elevated risk of overweight at age 18 regardless of their psychosocial risk, mental health/cognition, and pubertal development (Table 2– models 2, 3, and 4).

Does bullying victimization precede overweight?

At the time of bullying victimization (at ages 10 and 12 years), bullied children were not perceived by research workers to be more overweight than non-bullied children (Table 1). Similarly, bullied children did not show a higher risk of overweight, BMI, or waist-hip ratio at age 12, in a subsample (N=173) with anthropometric measures (Table 1). Furthermore, the association between bullying victimization and overweight at age 18 remained after accounting for childhood weight ratings (Table 2- model 5), as well as genetic risk of overweight and birth weight (Table 2- model 6).

Discussion

This cohort study showed that childhood bullying victimization is associated with overweight in young adulthood. First, we found some evidence of a *dose-response relationship*, in that the risk of being overweight increased as a function of the chronicity of bullying victimization in unadjusted analyses. Second, the association between bullying victimization and overweight was *consistent* across different measures of overweight, including categorical and continuous measures of BMI, and waist-hip ratio, a measure of central adiposity. Third, the findings were *specific* to bullying victimization by peers and not explained by co-occurring maltreatment by adults. Fourth, bullied children exhibited greater risk of overweight *independent* of potential confounders, such as socioeconomic status and food insecurity, child mental health/cognition, and pubertal development. Finally, the association was consistent with the hypothesized *temporal priority*, in that bullied children were not overweight at the time of victimization, but became overweight in young adulthood independent of (i) prior weight in childhood and (ii) pre-existing genetic and fetal liability.

Our findings should be considered in the context of some limitations. First, we studied a cohort of twins and our findings may not generalize to singletons. However, the prevalence of bullying and overweight in this sample is similar to that shown in studies of singletons (bullying prevalence: 42% by age 12 in E-Risk vs. 37% by age 13 in the Avon Longitudinal Study of Parents and Children (ALSPAC)(46); overweight prevalence at age 18: 23% in E-Risk vs. 23% in ALSPAC (47)). Second, we did not have anthropometric measures in childhood for the whole sample and relied on researcher workers' ratings of weight at ages 10 and 12, which may be liable to misclassification. However, the validity of these measures was supported by evidence that weight ratings at ages 10 and 12 (i) were correlated with body mass measured at age 12 in a subsample, and (ii) predicted overweight measures at age 18. Third, because we did not measure overweight throughout the observational period for bullying exposure, we cannot rule out the possibility that victims of

bullying were overweight at some point in childhood, as some (13, 21, 48) but not all (14) studies have shown. However, it is unlikely that reverse causation accounted for the findings, as bullied children became overweight at age 18 independent of childhood weight ratings and genetic and fetal liability to overweight. Fourth, unmeasured variables may have confounded the findings. Therefore, it is reassuring that our findings are consistent with experimental research from non-human primates (6, 7). Despite these limitations, our findings have implications for future research, clinical practice, and public health.

With regard to future research, studies should identify the mechanisms underlying the association between early life stress and overweight in later life. Our findings are consistent with the allostatic load theory prediction that more chronic exposure to psychosocial stress is associated with the greatest metabolic abnormalities (49). It is possible that early life stress could give rise to a 'thrifty' phenotype, characterized by high energy intake and/or low energy expenditure (8). For example, children exposed to early life stress may eat more, due to impaired inhibitory control over feeding linked to prefrontal cortex abnormalities (49). These children may also 'self-medicate' with high-calorie food to dampen chronic HPA axis activation (50). These hypotheses are consistent with evidence showing that childhood bullying victimization predicts bulimia and binge eating (51). Children exposed to early life stress may also expend less energy due to inflammation-related fatigue and physical inactivity (14, 52). In addition to biological explanations, social mechanisms may operate. For example, bullied children may avoid group sporting activities to reduce the risk of further victimization from peers. It is important to identify such mechanisms to inform the development of clinical interventions to prevent maltreated and bullied children from becoming overweight.

With regard to clinical practice, efforts should be made to support bullied children in order to prevent them from becoming overweight. It is possible that addressing unhealthy behaviors, such as comfort eating and physical inactivity (53), could help prevent bullied

children from becoming overweight. Such unhealthy behaviors might be partly linked to mental illness, and thus holistic approaches may bring the greatest benefits.

With regard to public health, our findings further highlight the importance of investing in anti-bullying interventions. Given the high prevalence of bullying and overweight, it is possible that effective anti-bullying strategies, such as targeted policies (11) and whole-school interventions (10), could help reduce the large public health burden due to overweight.

References

1. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011-2012. *Journal of the American Medical Association*. 2014;311:806-14.
2. Reilly JJ, Kelly J. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: systematic review. *Int J Obes*. 2011;35:891-8.
3. Morgan E, Dent M. The economic burden of obesity. Oxford: National Obesity Observatory, 2010.
4. Curioni CC, Lourenco PM. Long-term weight loss after diet and exercise: a systematic review. *Int J Obes Relat Metab Disord*. 2005;29:1168-74.
5. Dietz WH. Critical periods in childhood for the development of obesity. *Am J Clin Nutr*. 1994;59:955-9.
6. Kaufman D, Banerji MA, Shorman I, Smith EL, Coplan JD, Rosenblum LA, Kral JG. Early-life stress and the development of obesity and insulin resistance in juvenile bonnet macaques. *Diabetes*. 2007;56:1382-6.
7. Conti G, Hansman C, Heckman JJ, Novak MF, Ruggiero A, Suomi SJ. Primate evidence on the late health effects of early-life adversity. *Proc Natl Acad Sci U S A*. 2012;109:8866-71.
8. Danese A, Tan M. Childhood maltreatment and obesity: systematic review and meta-analysis. *Mol Psychiatry*. 2014;19:544-54.
9. Power C, Pinto Pereira SM, Li L. Childhood maltreatment and BMI trajectories to mid-adult life: Follow-up to age 50y in a British birth cohort. *PLoS One*. 2015;10:e0119985.
10. Vreeman RC, Carroll AE. A systematic review of school-based interventions to prevent bullying. *Arch Pediatr Adolesc Med*. 2007;161:78-88.
11. Hatzenbuehler ML, Schwab-Reese L, Ranapurwala SI, Hertz MF, Ramirez MR. Associations between antibullying policies and bullying in 25 states. *JAMA Pediatrics*. 2015;169:e152411.
12. Midei A, Matthews K. Interpersonal violence in childhood as a risk factor for obesity: a systematic review of the literature and proposed pathways. *Obesity Reviews*. 2011;12:e159-e72.
13. Qualter P, Murphy SM, Abbott J, Gardner KJ, Japel C, Vitaro F, Boivin M, Tremblay RE. Developmental associations between victimization and body mass index from 3 to 10 years in a population sample. *Aggressive Behav*. 2015;41:109-22.
14. Takizawa R, Danese A, Maughan B, Arseneault L. Bullying victimization in childhood predicts inflammation and obesity at mid-life: a five-decade birth cohort study. *Psychol Med*. 2015;45:1-11.
15. Lee CMY, Huxley RR, Wildman RP, Woodward M. Indices of abdominal obesity are better discriminators of cardiovascular risk factors than BMI: a meta-analysis. *J Clin Epidemiol*. 2008;61:646-53.
16. Bowes L, Arseneault L, Maughan B, Taylor A, Caspi A, Moffitt TE. School, neighborhood, and family factors are associated with children's bullying involvement: A nationally representative longitudinal study. *J Am Acad Child Adolesc Psychiatry*. 2009;48:545-53.
17. Cluver L, Bowes L, Gardner F. Risk and protective factors for bullying victimization among AIDS-affected and vulnerable children in South Africa. *Child Abuse Negl*. 2010;34:793-803.

18. Drewnowski A, Specter S. Poverty and obesity: the role of energy density and energy costs. *The American Journal of Clinical Nutrition*. 2004;79:6-16.
19. Craig WM, Pepler D, Connolly J, Henderson K, editors. Developmental context of peer harassment in early adolescence: The role of puberty and the peer group. New York: The Guilford Press; 2001.
20. Mamun A, Hayatbakhsh M, O'Callaghan M, Williams G, Najman J. Early overweight and pubertal maturation—pathways of association with young adults' overweight: a longitudinal study. *Int J Obes*. 2009;33:14-20.
21. Sweeting H, Wright C, Minnis H. Psychosocial correlates of adolescent obesity, 'slimming down' and 'becoming obese'. *J Adolesc Health*. 2005;37:409. e9-. e17.
22. Belsky DW, Moffitt TE, Houts R, Bennett GG, Biddle AK, Blumenthal JA, Evans JP, Harrington H, Sugden K, Williams B. Polygenic risk, rapid childhood growth, and the development of obesity: evidence from a 4-decade longitudinal study. *Arch Pediatr Adolesc Med*. 2012;166:515-21.
23. Oken E, Gillman MW. Fetal origins of obesity. *Obes Res*. 2003;11:496-506.
24. Trouton A, Spinath FM, Plomin R. Twins early development study (TEDS): a multivariate, longitudinal genetic investigation of language, cognition and behavior problems in childhood. *Twin Research*. 2002;5:444-8.
25. Moffitt TE, E-Risk Study Team. Teen-aged mothers in contemporary Britain. *J Child Psychol Psychiatry*. 2002;43:727-42.
26. Odgers CL, Caspi A, Russell MA, Sampson RJ, Arseneault L, Moffitt TE. Supportive parenting mediates neighborhood socioeconomic disparities in children's antisocial behavior from ages 5 to 12. *Dev Psychopathol*. 2012;24:705-21.
27. Odgers CL, Caspi A, Bates CJ, Sampson RJ, Moffitt TE. Systematic social observation of children's neighborhoods using Google Street View: a reliable and cost-effective method. *J Child Psychol Psychiatry*. 2012;53:1009-17.
28. Bowes L, Maughan B, Ball H, Shakoor S, Ouellet-Morin I, Caspi A, Moffitt TE, Arseneault L. Chronic bullying victimization across school transitions: the role of genetic and environmental influences. *Dev Psychopathol*. 2013;25:333-46.
29. Ronning J, Sourander A, Kumpulainen K, Tamminen T, Niemelä S, Moilanen I, Helenius H, Piha J, Almqvist F. Cross-informant agreement about bullying and victimization among eight-year-olds: whose information best predicts psychiatric caseness 10–15 years later? *Soc Psychiatry Psychiatr Epidemiol*. 2009;44:15-22.
30. Totura CMW, Green AE, Karver MS, Gesten EL. Multiple informants in the assessment of psychological, behavioral, and academic correlates of bullying and victimization in middle school. *J Adolesc*. 2009;32:193-211.
31. Shakoor S, Jaffee SR, Andreou P, Bowes L, Ambler AP, Caspi A, Moffitt TE, Arseneault L. Mothers and children as informants of bullying victimization: results from an epidemiological cohort of children. *Journal of abnormal child psychology*. 2011;39:379-87.
32. Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z, Wei R, Curtin LR, Roche AF, Johnson CL. 2000 CDC Growth Charts for the United States: methods and development. *Vital and health statistics Series 11, Data from the national health survey*. 2002:1-190.
33. Jaffee SR, Bowes L, Ouellet-Morin I, Fisher HL, Moffitt TE, Merrick MT, Arseneault L. Safe, stable, nurturing relationships break the intergenerational cycle of abuse: A prospective nationally representative cohort of children in the United Kingdom. *J Adolesc Health*. 2013;53:S4-S10.

34. Danese A, Dove R, Belsky DW, Henchy J, Williams B, Ambler A, Arseneault L. Leptin deficiency in maltreated children. *Translational Psychiatry*. [Original Article]. 2014;4:e446.
35. Dodge KA, Bates JE, Pettit GS. Mechanisms in the cycle of violence. *Science*. 1990;250:1678-83.
36. Trzesniewski KH, Moffitt TE, Caspi A, Taylor A, Maughan B. Revisiting the association between reading achievement and antisocial behavior: New evidence of an environmental explanation from a twin study. *Child Development*. 2006;77:72-88.
37. Melchior M, Caspi A, Howard LM, Ambler AP, Bolton H, Mountain N, Moffitt TE. Mental health context of food insecurity: a representative cohort of families with young children. *Pediatrics*. 2009;124:e564-e72.
38. Achenbach T. Manual for the Child Behaviour Checklist and 1991 profile. Burlington, University of Vermont. 1991.
39. Achenbach T. Manual for the Teacher's Report Form and 1991 Profile. Burlington, VT. 1991.
40. Sattler JM. Assessment of children: WISC—III and WPPSI—R supplement: Jerome M. Sattler; 1992.
41. Wechsler D. Wechsler preschool and primary scale of intelligence-revised: Psychological Corporation; 1989.
42. Tanner JM, Whitehouse RH. Clinical longitudinal standards for height, weight, height velocity, weight velocity, and stages of puberty. *Archives of disease in childhood*. 1976;51:170-9.
43. Kendler KS, Kessler RC, Walters EE, MacLean C, Neale MC, Heath AC, Eaves LJ. Stressful life events, genetic liability, and onset of an episode of major depression in women. *Am J Psychiatry*. 1995;152:833-42.
44. Arseneault L, Cannon M, Fisher HL, Polanczyk G, Moffitt TE, Caspi A. Childhood trauma and children's emerging psychotic symptoms: a genetically sensitive longitudinal cohort study. *Am J Psychiatry*. 2011;168:65-72.
45. Tully LA, Arseneault L, Caspi A, Moffitt TE, Morgan J. Does maternal warmth moderate the effects of birth weight on twins' attention-deficit/hyperactivity disorder (ADHD) symptoms and low IQ? *J Consult Clin Psychol*. 2004;72:218-26.
46. Lereya ST, Copeland WE, Costello EJ, Wolke D. Adult mental health consequences of peer bullying and maltreatment in childhood: two cohorts in two countries. *Lancet Psychiatry*. 2015:1-8.
47. Johnson W, Li L, Kuh D, Hardy R. How has the age-related process of overweight or obesity development changed over time? Co-ordinated analyses of individual participant data from five United Kingdom birth cohorts. *PLoS Med*. 2015;12:e1001828.
48. Mamun AA, O'Callaghan MJ, Williams GM, Najman JM. Adolescents bullying and young adults body mass index and obesity: a longitudinal study. *International Journal of Obesity*. [Original Article]. 2013;37:1140-6.
49. Danese A, McEwen BS. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiol Behav*. 2012;106:29-39.
50. Dallman MF, Pecoraro N, Akana SF, La Fleur SE, Gomez F, Houshyar H, Bell M, Bhatnagar S, Laugero KD, Manalo S. Chronic stress and obesity: a new view of "comfort food". *Proc Natl Acad Sci U S A*. 2003;100:11696-701.
51. Copeland WE, Bulik CM, Zucker N, Wolke D, Lereya ST, Costello EJ. Does childhood bullying predict eating disorder symptoms? A prospective, longitudinal analysis. *Int J Eat Disord*. 2015.

52. Copeland WE, Wolke D, Lereya ST, Shanahan L, Worthman C, Costello EJ. Childhood bullying involvement predicts low-grade systemic inflammation into adulthood. *Proc Natl Acad Sci U S A*. 2014;111:7570-5.
53. Storch EA, Milsom VA, DeBraganza N, Lewin AB, Geffken GR, Silverstein JH. Peer victimization, psychosocial adjustment, and physical activity in overweight and at-risk-for-overweight youth. *J Pediatr Psychol*. 2007;32:80-9.

Table 1. Participant characteristics according to childhood bullying victimization

		Childhood bullying victimization ^a			<i>p</i> -value*
		None (n=1,255)	Transitory (n=605)	Chronic (n=286)	
<i>Measures of overweight at age 18</i>					
Overweight, n (%)		233 (20.09)	140 (25.04)	74 (28.79)	0.002
BMI, kg/m ²		22.76 (0.17)	23.23 (0.27)	23.83 (0.38)	0.004
Waist-hip ratio		0.81 (0.002)	0.82 (0.004)	0.83 (0.005)	<0.001
<i>Measures of overweight in childhood</i>					
Overweight (age 12) ^b , n(%)		28 (35.90)	24 (44.44)	12 (29.27)	0.66
BMI (age 12) ^b , kg/m ²		20.62 (0.51)	21.35 (0.71)	20.57 (0.91)	0.91
Waist-hip ratio (age 12) ^b		0.84 (0.01)	0.84 (0.01)	0.83 (0.01)	0.57
Weight rating (age 12) ^c		3.98 (0.03)	4.07 (0.05)	4.01 (0.07)	0.30
Weight rating (age 10) ^c		3.90 (0.04)	3.88 (0.06)	3.84 (0.08)	0.49
<i>Covariate</i>					
Female sex, n (%)		685 (54.58)	288 (47.60)	129 (45.10)	0.004
Ethnicity, n (%)		102 (8.13)	28 (4.63)	8 (2.80)	0.002
Genetic risk of overweight		0.88 (0.03)	0.97 (0.06)	1.01 (0.08)	0.039
Birth weight (kg)		2.44 (0.02)	2.42 (0.03)	2.46 (0.04)	0.86
Child internalizing problems		14.06 (0.31)	15.21 (0.50)	18.77 (0.76)	<0.001
Child externalizing problems		16.25 (0.43)	19.58 (0.74)	24.94 (1.16)	<0.001
Child IQ		97.00 (0.49)	96.02 (0.79)	90.11 (1.02)	<0.001
Child SES, n (%)	High	472 (37.61)	187 (30.91)	65 (22.73)	<0.001
	Medium	417 (33.23)	201 (33.22)	84 (29.37)	
	Low	366 (29.16)	217 (35.87)	137 (47.90)	
Food insecurity, n (%)	None	1,129 (89.96)	505 (83.47)	234 (81.82)	<0.001
	Episodic	99 (7.89)	67 (11.07)	38 (13.29)	

	Sustained	27 (2.15)	33 (5.45)	14 (4.90)	<0.001
Child maltreatment, n (%)	None	1,046 (83.35)	445 (73.55)	194 (67.83)	
	Probable	174 (13.86)	117 (19.34)	45 (15.73)	
	Definite	35 (2.79)	43 (7.11)	47 (16.43)	<0.001
Pubertal development		4.41 (0.06)	4.48 (0.10)	4.65 (0.14)	0.10

Results are presented as means and standard errors unless otherwise stated.

*Associations with continuous variables were estimated with linear regression models and associations with categorical variables were estimated with logistic regression models adjusted for familial clustering.

^a Childhood bullying victimization was assessed between ages 5-12 years.

^b At the age-12 follow-up, research workers took anthropometric measurements in a subsample of 173 study members (no victimization: n=78; transitory victimization: n=54; chronic victimization: n=41). The cut-off for overweight at 12 years was BMI \geq 21 for males and BMI \geq 21.7 for females, in accordance to 85th percentile from CDC growth charts (32)

^c Research workers rated study members' weights on a scale from 1 (underweight) to 7 (overweight)

Table 2. Association between childhood bullying victimization and measures of overweight at age 18. All models adjust for gender and ethnicity.

	Baseline	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
	(unadjusted)	(adjusted for maltreatment)	(adjusted for SES and food insecurity)	(adjusted for child mental health/ cognition)	(adjusted for pubertal development)	(adjusted for child weight ratings)	(adjusted for genetic and fetal liability)	(adjusted for all covariates)
Overweight								
None	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Transitory	1.37 (1.06;1.78)	1.36 (1.05;1.77)	1.28 (0.98;1.66)	1.29 (0.99;1.68)	1.34 (1.03;1.74)	1.40 (1.04;1.88)	1.31 (1.01;1.71)	1.27 (0.94;1.73)
Chronic	1.69 (1.21;2.35)	1.63 (1.16;2.87)	1.51 (1.08;2.12)	1.44 (1.01;2.04)	1.59 (1.13;2.24)	1.95 (1.31;2.90)	1.62 (1.16;2.26)	1.65 (1.09;2.49)
BMI								
None	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Transitory	0.51 (-0.02;1.03)	0.48 (-0.05;1.01)	0.37 (-0.15;0.89)	0.34 (-0.19;0.88)	0.45 (-0.07;0.97)	0.33 (-0.09;0.76)	0.28 (-0.15;0.72)	0.12 (-0.26;0.50)
Chronic	1.12 (0.37;1.87)	1.05 (0.29;1.80)	0.91 (0.17;1.65)	0.70 (-0.10;1.50)	0.99 (0.25;1.73)	1.19 (0.62;1.76)	0.77 (0.12;1.42)	0.73 (0.18;1.29)
Waist hip ratio								
None	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Transitory	0.70 (0.01;1.40)	0.65 (-0.05;1.35)	0.56 (-0.14;1.26)	0.50 (-0.19;1.19)	0.67 (-0.02;1.37)	0.57 (-0.06;1.21)	0.52 (-0.12;1.17)	0.34 (-0.27;0.96)
Chronic	1.76 (0.84;2.69)	1.64 (0.72;2.57)	1.46 (0.53;2.39)	1.16 (0.22;2.11)	1.70 (0.77;2.62)	1.80 (0.90;2.69)	1.48 (0.60;2.35)	1.17 (0.28;2.06)

Results are presented as odds ratios (for overweight) or unstandardized regression coefficients (for BMI and waist-hip ratio) with 95% confidence intervals. Values for waist-hip ratio were multiplied by 100 for these analyses.

Data on covariates were missing from between <1% (child IQ, food insecurity, and weight rating at age 12) to 7% (birth weight), with an average of 2.9% missing. We replaced missing values for covariates with means of the observed data, resulting in N=1,976 for analyses on overweight and BMI, and N=1,987 for analyses on waist-hip ratio. Sensitivity analyses showed similar findings regardless of whether we replaced missing data, conducted a complete case analysis, or used listwise deletion.

Table 3. Association between covariates with measures of overweight at age 18.

Covariate	Level	Measures of overweight at age 18					
		Overweight		BMI		Waist-hip ratio	
		OR	<i>p</i> -value	b	<i>p</i> -value	b	<i>p</i> -value
Female sex		1.45 (1.13;1.86)	0.003	0.57 (0.03;1.10)	0.038	-3.26 (-3.89;-2.63)	<0.001
Ethnicity		0.99 (0.58;1.68)	0.97	-0.24 (-1.60;1.12)	0.73	-0.82 (-2.21;0.58)	0.25
Genetic risk		3.01 (2.55;3.55)	<0.001	2.41 (2.14;2.68)	<0.001	1.79 (1.45;2.13)	<0.001
Birth weight (kg)		1.22 (0.95;1.56)	0.12	0.51 (0.06;0.96)	0.025	0.23 (-0.37;0.83)	0.45
Child internalizing problems		1.01 (0.99;1.02)	0.40	0.01 (-0.02;0.03)	0.51	0.01 (-0.02;0.04)	0.45
Child externalizing problems		1.02 (1.01;1.02)	<0.001	0.04 (0.02;0.06)	<0.001	0.08 (0.06;0.11)	<0.001
Child IQ		0.99 (0.98;1.00)	0.007	-0.03 (-0.04;-0.01)	0.002	-0.05 (-0.07;0.03)	<0.001
Childhood SES	High	Ref		Ref		Ref	
	Medium	1.65 (1.21;2.26)	0.002	0.95 (0.33;1.57)	0.003	1.24 (0.46;2.02)	0.002
	Low	1.82 (1.33;2.49)	<0.001	1.18 (0.53;1.83)	<0.001	2.00 (1.18;2.82)	<0.001
Food insecurity	None	Ref		Ref		Ref	
	Episodic	1.48 (0.99;2.19)	0.054	0.55 (-0.37;1.47)	0.24	1.16 (0.04;2.29)	0.042
	Sustained	2.13 (1.13;4.01)	0.020	1.61 (-0.03;3.26)	0.06	0.70 (-1.42;2.82)	0.52
Child maltreatment	None	Ref		Ref		Ref	

Child bullying predicts overweight in young adulthood

	Probable	0.90 (0.65;1.26)	0.55	0.03 (-0.66;0.72)	0.93	0.50 (-0.34;1.35)	0.25
	Definite	1.46 (0.88;2.42)	0.15	0.71 (-0.38;1.79)	0.20	1.60 (0.21;2.99)	0.024
Weight rating at age 10		3.31 (2.81;3.89)	<0.001	2.31 (2.08;2.55)	<0.001	1.66 (1.36;1.97)	<0.001
Weight rating at age 12		4.21 (3.47;5.11)	<0.001	2.92 (2.63;3.21)	<0.001	2.27 (1.90;2.63)	<0.001
Pubertal development		1.22 (1.14;1.30)	<0.001	0.39 (0.26;0.53)	<0.001	0.04 (-0.15;0.22)	0.70

Results are presented as odds ratios (for overweight) or unstandardized regression coefficients (for BMI and waist-hip ratio) with 95% confidence intervals. Values for waist-hip ratio were multiplied by 100 for these analyses.

Figure 1. The association between childhood bullying and overweight at age 18. (a) Percentage overweight at age 18 (and SE) according to bullying victimization. (b) Median, range, and interquartile range of BMI at age 18 according to bullying victimization. Individual data points are displayed. (c) Median, range, and interquartile range of waist-hip ratio at age 18 according to bullying victimization. Individual data points are displayed.

